

symptoms seemed not to have been suspected. I studied such a patient with the view of unmasking his hyperopia, only to find at the next test that his hyperopia was considerably higher than I had suspected at the first visit, and often to find it at a subsequent test still higher. The statement which I made 14 years ago, with regard to the latency of hyperopia deserves still to be emphasized: "That it is not more frequent or proportionately greater in childhood, or in early adult than in middle life." I believe now that latent hyperopia is more frequent and more troublesome after 40 than before that age. Hyperopia of low degree is more apt to be latent after 50, and at that age is very often the cause of eye-strain of obscure origin.

The age at which accommodation ceases or becomes inappreciable is seen to vary widely. In one case it had entirely disappeared at the age of 46; in the majority of cases it was absent or reduced to 0.25 D. after 60; but in one case there remained 1.50 D. at the age of 68 years, which would seem to indicate that it may persist to the age of 75 or 80.

In taking the near point of old people, it is necessary to guard against two important sources of error. The smallness of the pupil may enable the patient to read quite fine print without having his eye optically adjusted to focus accurately for the distance at which it is read, or the refraction may vary in different parts of the pupil, so that good distant vision may be obtained through peripheral parts of the pupil, while the central part of the pupil (or the whole of its area when the pupil is contracted with strong convergence) may be decidedly myopic. The former may be guarded against by noting that the pupil is large, or even by dilating it with cocaine. The latter may be excluded by skiascopic examination. In the more remarkable cases with which I have met I am sure that these sources of error have been excluded.

It is clear that it is not unusual for the accommodation to persist to a considerably greater age than we have been accustomed to suppose. And I know of no reason why patients with healthy eyes who still possess accommodation of practical importance should not be allowed to use it. They generally appreciate the increased region of distinct vision which the weaker lenses afford them.

**Sex Difference in Accommodation**—Another point which is of some interest, brought out by this study, and which was a surprise to me, was the difference in accommodative power between the two sexes. Perhaps none of us would be surprised that women giving their ages between 30 and 50 should show less accommodation than men between the same ages. I have tried to guard against this by excluding from my tables a great many women between these ages in whom the statement of age was decidedly open to suspicion. I also tested my tables by comparison with the accommodative power of a smaller series of women whose ages were positively known to me; and I think the differences shown in table III really exist:

TABLE III.

Age.	Male.	Female.	Age.	Male.	Female.
5 to 10...	12.44	12.13	35 to 40...	5.89	5.62
10 to 15...	10.87	10.58	40 to 45...	4.49	4.30
15 to 20...	9.91	9.32	45 to 50...	2.89	2.78
20 to 25...	8.89	8.58	50 to 55...	1.53	1.48
25 to 30...	8.	7.80	55 to 60...	0.90	0.83
30 to 35...	6.87	6.73	Over 60...	0.65	0.50

The surprising thing is that these differences were found to exist from early childhood until the final decline of accommodation.

**Inequality of the Two Eyes**—The frequency of inequality of accommodative power in the two eyes is not brought out by these tables. A moderate inequality, 1 D. or less, is not rare. But in persons who have accommodation of 5 D. or over this difference is of little practical importance. With the advent of presbyopia moderate differences between the accommodative power of the two eyes more frequently need to be considered in the relief of eye-strain. Of course the difference between the strength of the two lenses given for presbyopia will not equal the differences between the accommodative powers of the two eyes. Commonly the difference between the lenses will be the same proportion of the difference of accommodative power, as the proportion of accommodation habitually to be used for near work; or rather less than this. Commonly the difference between the lenses will be from one-third to two-thirds the difference of accommodation, or in young persons even less than one-third. Higher differences of accommodative power are clearly due to unilateral or unequal pareses of the ciliary muscle, which are almost always accompanied by similar pareses of the sphincter of the pupil. But the low degrees exist without inequality of the pupils.

In presenting this study of the amplitude of accommodation, let me especially call your attention to

The wide range of variation in persons of the same age and apparently healthy eyes, making the near point a poor indication of the amount of hyperopia latent.

The frequency and importance of subnormal accommodation, even in childhood.

The variability of the age at which presbyopia begins.

The frequency of hyperopia latent after fifty.

The practical importance of differences in accommodative power between the two eyes.

And in general, the urgent necessity for studying carefully the amplitude of accommodation in every case of eye-strain.

### ANEURISM OF THE LEFT VENTRICLE WITH REPORT OF CASE.\*

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Cardiac aneurisms are of very unusual occurrence, and as such should be looked upon principally as pathological curiosities. By this I mean that they are seldom or never clinically recognized, being

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found as a general rule only at autopsy. This may be accounted for by the fact that such aneurisms are usually occasioned by myocarditic or atheromatous trouble, and that the clinical signs and symptoms of the former are those of the latter disease.

Occasionally a cardiac aneurism may attain an unusual size and cause pressure symptoms in the mediastinum, in which case a radiogram may reveal its presence. However, such an occurrence is so rare that it may be dismissed with a word. Our leading medical authorities, such as Osler (1), Anders (2), Butler (3), Musser (4), Albutt (5), Strumpell (6), Leube (7), and Krehl (8), make identically similar statements: that the condition cannot be diagnosed clinically with any degree of certainty, and that it is most often caused by fibroid changes in the heart wall, due to a block of the coronary arteries, the latter occasioned by atheromatous degeneration and sclerotic patches.

Searching through the literature I can only find the record of one case which was diagnosed clinically, that of Vollker (9), quoted by G. Hall in his article in the *Edinburgh Journal*, October, 1902. In all probability behind this atheroma, syphilis acts as the prevailing etiological factor. Less frequently aneurism of the heart wall is caused by anemic infarcts fatty degeneration, mural endocarditis, and wounds of the heart. There is a case on record, published by Commotti (10) and Vittorio, of a basal heart aneurism of gummatous origin. This, to be sure, it is needless to remark, is of unusual occurrence. We find the condition more in males than in females, due, according to Strumpell, to the more frequent occurrence in them of coronary sclerosis.

Hendys, in a total of 208 collected cases, reports the occurrence of 74 per cent in males and 26 per cent in females. Three forms of cardiac aneurisms are usually described: First, of the coronary arteries; second, of the valves; third, of the heart wall. Coronary and valvular aneurism seldom occurs, the common location being the heart wall. Most authorities give the usual site of a cardiac aneurism as the lower part of the left ventricle, near the apex. Sir R. Douglas Powell (11) states that it occurs near the apex in 59 per cent in all cases, Von Jirgensen (12), in the Nothnagle Series, grants that heart aneurisms occur very seldom in other places than near the apex.

Hall collected 112 cases, the site of the aneurism being as follows:

Left ventricle, 92 cases; right ventricle, 1 case; left auricle, 2 cases; ventricular septum (a) muscular part, 8 cases; (b) membranous part, 7 cases.

The literature of cardiac aneurism, although quite abundant, has not been entirely at my disposal, owing to our still limited library facilities in San Francisco, but I have been able to ascertain that up to 1902 the number of recorded cases of cardiac aneurism was about 300. To this contribution of 300 case the most notable was by Legg (13), in 1883, who, in his famous Bradshaw lecture, presented a collection of 90 cases. John Lindsay Stevens (14), presented a collection in 1894 of 21 cases, in which in 16 he demonstrated how aneurism of the

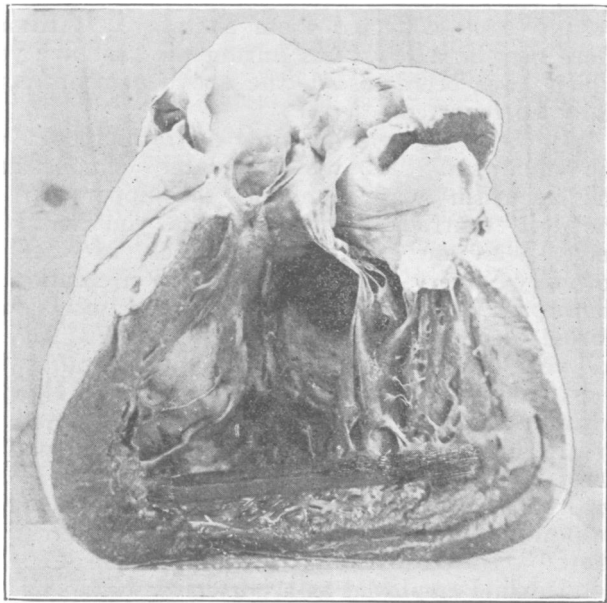
heart wall with thrombosis may follow a calcareous infiltration or obstruction of a coronary artery, and in which he gave forth the very plausible theory that the development of fibrous patches in the heart wall, "probably affords the structural basis of a very large number of cases of angina pectoris." Since 1902 I have made a very careful search of all the literature at my command, and find the following cases have been reported:

Two cases by Benedict of Budapest (15); two cases by Davidsohn and Strauss, Berlin (16); one case by D. G. Hall, Edinburgh (17); one case by Wadsworth, Philadelphia (18); one case by M. W. Hall, Chicago (19); two cases by Bourland (20); two cases by Herczel (21); two cases by Gruner (22); one case by Roffo (23); one case by Goettsch (24); one case by Marie (25); two cases by Potts (26); one case by Loraine (27); one case by M. Riehl (28); making a total of 20 cases in five years, or 320 cases collected from literature. Although there may be a few cases during the past five years which have been missed, the above presents essentially all reported.

My own case, which I now desire to report, was the aneurism. As will be later demonstrated, the aneurism was found in the upper part of the left ventricle. Its cause, as you will perceive, was an atheromatous block of the left coronary artery cutting off the circulation to that part of the heart wall, and occasioning fibroid degeneration. The patient, W. A., age 62 years, a salesman by occupation, came into the medical service of Mt. Zion Hospital March 19th, 1907, complaining of shortness of breath, fainting spells, pains in the feet, especially across the instep and inner side of the foot, and restlessness during sleep. Also occasional attacks of palpitation and a feeling of tightness across the chest. His past history shows that in October, 1906, while employed as a porter, he began to have difficulty in breathing, which necessitated his giving up his work. Some little time later he began to have precordial pains after eating. A week ago patient began to have attacks of vertigo and fainting, which have increased in frequency and which are produced by any kind of exertion. From October, 1906, to February 2nd, 1907, he was treated by a number of physicians for heart trouble, and was aspirated for ascites. At this time he also had swelling of the eyelids. Seven or eight years ago had difficulty in holding his urine, but no trouble at present. Admits lues as a young man and several attacks of gonorrhœa. Family history unimportant. Previously to present illness was always a well man, and up to October, 1906, always worked at his occupation without intermission.

Status: Well built, well nourished man, weight 165 pounds, no glandular enlargements, tongue slightly coated, pupils react normally to light and accommodation, no disturbance in the course of the cerebral nerves. Patient lies in bed with rather an anxious look on his face, and respiration somewhat labored. Chest well formed, moves equally and freely upon respiration. At both bases of the lungs percussion note is dull and breath sounds dimin-

ished; otherwise negative findings. Heart, apex beat hardly palpable, just outside the nipple line in the fifth interspace; borders: upper, upper third rib, left: one finger breadth, outside the nipple line, right: mid-sternal line; heart action is arrhythmic; sounds not very loud, and at apex is heard a soft blowing distant systolic murmur. Over the pulmonary and aortic orifices the sounds are pure. Pulse 90, irregular, intermittent, not very full. Abdomen slightly distended, liver extends below the margin of the ribs, the edge is hard; spleen is negative. No edema of the legs. Urine examination, color amber, acid 1022, albumen 0.05%, sugar negative. Microscopical examination negative; blood examination: Rbc., 4,500,000; wbc., 7,600; h., 90%.



The clinical diagnosis was as follows: Mitral insufficiency and myocarditis with an acute break in compensation. He was placed upon infus. digitalis 15.0 q. i. d. and aspirin 0.6 every two hours, and showed a daily improvement. March 24th his pulse was regular, quite full, 80 to the minute, and his heart sounds much louder and better in character than at any previous examination; his dyspnoea had vanished, and in all respects he felt very much improved. Late in the afternoon of this day he began to have an attack of tightness across the chest and pains in the precordial region, attempted to get out of bed, but fell back cyanotic and gasping, expiring shortly afterwards.

Autopsy performed by Dr. J. Schwarz, pathologist to Mt. Zion Hospital. Fairly well developed, well nourished middle aged man, no enlarged superficial glands, position of abdominal organs normal, the liver normal in size and position; slightly cyanotic, stomach, transverse colon and mesentery and omentum, normal, no fluid present in abdomen, peritoneum normal. Diaphragm reached to fifth intercostal space on either side; thorax well arched, symmetrical; right pleural cavity contained 1000 c. c. of straw-colored fluid;

right lung collapsed, otherwise negative; left pleural cavity contained 600 c. c. of straw-colored fluid, lung negative; pericardium contained 350 c. c. of straw-colored fluid. Heart: in general somewhat enlarged, the usual external tests show no insufficiency of the valve, heart measurements show length 14 cm., breadth 11 cm., thickness 7 cm. showing a moderate hypertrophy. The cut surface of the ventricular wall measures 2 cm., showing considerable hypertrophy. The right ventricle is dilated, flabby. Distributed through the walls of the left ventricle are numerous scars, especially in the papillary muscles. On the external surface of the left ventricle in the upper third is a thinned out area about three and a half cm. in diameter. The valves are all normal except the mitral, which contains a few thickenings. Beginning 8 cm. from the apex, just under the chordæ tendinæ is a cavity with a fibrous rim 4 cm. in diameter and 1½ cm. deep, containing a large clot. The clot removed shows the base of the cavity to correspond to the thinned out area on the external surface of the left ventricle. Small atheromatous patches are seen upon the aorta, the right coronary artery is atheromatous, the left likewise, and near its ending leading to the thinned out area on the ventricular wall is a calcareous patch almost completely blocking the artery. (This is, no doubt, the direct cause of the fibroid degeneration of the heart wall in this region, and the subsequent aneurism.) Spleen normal in size, slightly cyanotic; kidneys normal, except for slight cyanosis, pancreas normal. Pathological diagnosis: Hydrothorax pericarditis, mitral insufficiency, hypertrophy of left ventricle, dilation of right ventricle, aneurism of the left ventricle in the upper third near the auricle.

I have not aimed to add anything to the clinical diagnosis of cardiac aneurism by the presentation of this case. I have simply reported it for its pathological interest, and its unusual location.

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